

Perspective

Evaluating and Managing Cardiovascular Disease Risk Factors in HIV-Infected Patients

The primary risk factors for cardiovascular disease (CVD) in patients with HIV infection are the same as those for the general population. Some antiretroviral drugs are associated with a small increase in short-term risk of CVD that may become greater over longer periods of exposure. However, the absolute risk associated with use of these drugs is of greatest importance for patients already at moderate or high risk of CVD. The guiding principle in managing CVD risk for HIV-infected patients is to maintain control of the HIV infection while addressing the metabolic abnormalities that increase CVD risk. Lipid-lowering therapy with statins is very effective in reducing CVD risk, with levels of low-density lipoprotein cholesterol and non-high-density lipoprotein cholesterol constituting the primary treatment targets for most patients with dyslipidemia. This article summarizes a lecture by James H. Stein, MD, at the International AIDS Society–USA continuing medical education program held in Washington, DC, in June 2010.

Many HIV-infected patients now live long enough to acquire the diseases common in the HIV-seronegative population in the United States, such as cardiovascular disease (CVD). Special consideration needs to be given to predicting and managing CVD risk in the aging HIV-infected population.

Predicting Coronary Heart Disease Risk in the General Population

Along with aging, male sex, and a family history of premature heart disease, modifiable causes of coronary heart disease (CHD) in the general population include smoking, high blood pressure, high cholesterol levels, and diabetes mellitus. Data from 21 years to 30 years of follow-up from 3 large epidemiologic studies—the Chicago Heart Association Study, Framingham Heart Study, and MRFIT (Multiple Risk Factor Intervention Trial)—have shown that 85% to 100% of young and middle-

aged men and women who develop CHD have at least 1 established CHD risk factor, and 96% to 100% of persons in whom CHD develops have at least 1 risk factor above optimal levels (ie, total cholesterol level of 200 mg/dL or greater or use of cholesterol medication; systolic blood pressure [BP] greater than 120 mm Hg, diastolic BP greater than 80 mm Hg, or use of BP medication; current cigarette use; or diabetes mellitus) (Greenland et al, *JAMA*, 2003).

In the large, ongoing international INTERHEART study, comparisons between 52,000 myocardial infarction (MI) cases and 52,000 control subjects showed that 90% of the population's attributable risk was accounted for by 9 risk factors: high lipid levels, smoking, diabetes mellitus, and hypertension, as well as inadequate consumption of fruits and vegetables, lack of adequate exercise, excessive alcohol consumption, abdominal obesity, and high levels of psychosocial stress. A noteworthy finding was that modifiable lifestyle risk factors accounted for 63% of attributable risk (Yusuf et al, *Lancet*, 2004). Thus, in 2010, when such risk factors are highly prevalent in the general population, the problem in predicting CHD is one of specificity—separating out the people with

risk factors who will experience a CVD event from those with risk factors who will not.

Predicting Coronary Heart Disease Risk in the HIV-Infected Population

Similar considerations apply with regard to CHD risk in the HIV-infected population. The D:A:D (Data Collection on Adverse Events of Anti-HIV Drugs) study showed that a statistically significant increased risk of MI was associated with such traditional risk factors as increasing age, male sex, family history of heart disease, current and former smoking, and a prior CVD event, with current smoking (relative risk [RR], 2.83) and prior CVD event (RR, 4.30) having the highest RRs. In addition, 5-year exposure to protease inhibitor (PI) therapy was a statistically significant risk factor (RR, 2.01 per 5 years) (D:A:D Study Group, *N Engl J Med*, 2007). However, the primary analysis of the D:A:D study omitted diabetes mellitus, hypertension, and total and high-density lipoprotein cholesterol (HDL-C) levels from the risk models. When these factors were included, all had a substantial effect in predicting MI; the RR associated with PI treatment remained statistically significant but was reduced to 1.61 per 5 years of exposure, lower than that associated with prior cardiovascular event (RR, 4.64), male sex (RR, 2.13), diabetes mellitus (RR, 1.86), and current (RR, 2.92) and former (RR, 1.63) smoking.

Despite the fact that some antiretroviral drugs may pose longer-term risks of CVD via metabolic effects, it is clear that antiretroviral therapy has had a profound effect in reducing all-cause mortality in HIV-infected patients with no discernible increase in short-term risk of CVD events or death. For example, a large retrospective study showed

Dr Stein is professor of medicine and director of preventive cardiology at the University of Wisconsin School of Medicine and Public Health.

See page 154 regarding CME credit for this article.

that between 1996 (the advent of the potent antiretroviral therapy era) and 2001, all-cause mortality declined dramatically, but there was no discernible increase in rates of death from CVD or cerebrovascular disease, hospital admission for CVD or cerebrovascular disease, or hospital admission for CVD compared with prior years (Bozzette et al, *N Engl J Med*, 2003).

Indeed, a recent study suggests that antiretroviral therapy may reduce CVD risk over the short term by improving endothelial function. In that study, 82 treatment-naïve, HIV-infected patients were randomly assigned to receive a nucleoside analogue reverse transcriptase inhibitor (nRTI)-sparing regimen (efavirenz plus ritonavir-boosted [r] lopinavir), a PI-sparing regimen (efavirenz plus nRTIs), or a nonnucleoside analogue reverse transcriptase inhibitor (NNRTI)-sparing regimen (lopinavir/r plus nRTIs) for 24 weeks. Brachial artery flow-mediated dilation, a measure of endothelial function, increased by 1.48% ($P < .001$) in all subjects considered together, and similar improvements were observed in each group despite substantial differences in lipid changes among the groups (Torriani et al, *J Am Coll Cardiol*, 2008).

The findings indicating that control of HIV viremia can improve endothelial function are consistent with CVD risk data from the SMART (Strategies for Management of Antiretroviral Therapy) trial, which compared a drug-conservation strategy based on CD4+ cell count (off treatment when CD4+ cell count $> 350/\mu\text{L}$ and resume treatment when count falls $< 250/\mu\text{L}$) with a continuous-viral-suppression strategy. Overall, patients in the drug-conservation group had a statistically significantly higher risk of death (RR, 2.6) than did patients in the viral-suppression group.

With regard to CVD events, the hazard ratios (HRs) for MI, percutaneous coronary intervention, or CVD death (HR, 1.57; $P = .05$) plus (inclusive of preceding endpoint) peripheral vascular disease, congestive heart failure, or coronary artery disease requiring medication (HR, 1.49; $P = .03$), plus (inclusive of preceding 2 endpoints) unobserved death of unknown cause

(HR, 1.58; $P = .009$) were substantially higher in the drug-conservation group than in the viral-suppression group. CVD events were not associated with being off antiretroviral therapy or with viral load. The ratio of total cholesterol level to HDL-C level was higher in the drug-conservation group because of a decrease in the level of HDL-C (Phillips et al, *Antivir Ther*, 2008; SMART Study Group et al, *N Engl J Med*, 2006).

Nevertheless, some antiretroviral drugs have been associated with increased risk of MI. A recent analysis from the D:A:D study assessed risk of MI associated with use of individual antiretroviral drugs (Worm et al, *J Infect Dis*, 2010). Overall, MI occurred in 580 of 33,308 patients. Among PIs, in-

dinavir and lopinavir/r (both of which are associated with increased metabolic abnormalities) were associated with a statistically significant increase in RR of MI per year of exposure (Figure 1). No increase in RR was observed with the NNRTIs nevirapine or efavirenz.

Among nRTIs, didanosine and abacavir were associated with a statistically significant increase in RR of MI for recent or current use, and abacavir was associated with a statistically significant increase in the RR per year of use. An analysis of patients receiving continuous antiretroviral therapy in the SMART study also showed that abacavir (but not didanosine) was associated with a higher risk of CVD than that of other nRTIs, with adjusted HRs

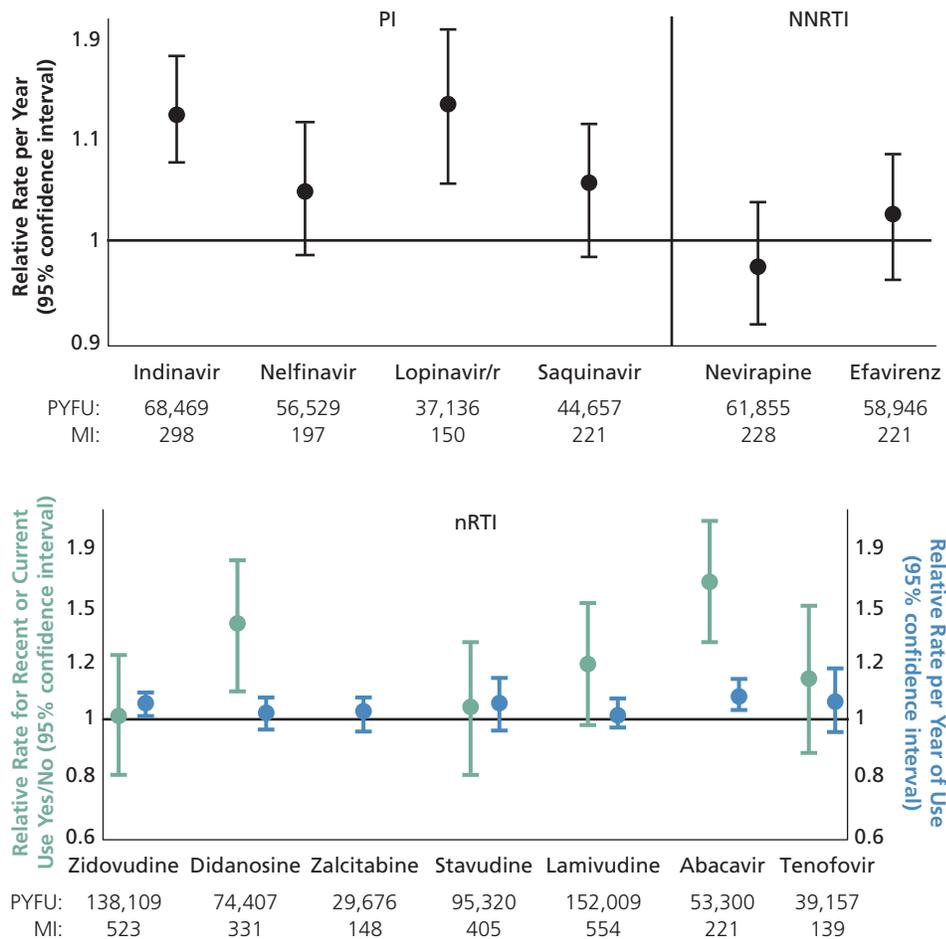


Figure 1. Top, Relative rate of myocardial infarction (MI) per year associated with protease inhibitor (PI) and nonnucleoside analogue reverse transcriptase inhibitor (NNRTI) use in the D:A:D (Data Collection on Adverse Events of Anti-HIV Drugs) study. Bottom, Relative rate of MI according to recent or current use and relative rate per year of use associated with nucleoside analogue reverse transcriptase inhibitor (nRTI) use in the D:A:D study. PYFU indicates patient-years of follow-up; /r, ritonavir boosted. Adapted from Worm et al, *J Infect Dis*, 2010, and Lundgren et al, *CROI*, 2009.

for abacavir use of 4.3 for MI, 1.8 for major CVD, and 1.9 for CVD using an expanded definition (SMART/INSIGHT and D:A:D Study Groups, *AIDS*, 2008). Abacavir also was associated with increased levels of high-sensitivity C-reactive protein and interleukin-6.

In summary, integrating the effects of traditional CVD risk factors and antiretroviral therapy–associated risk factors in HIV-infected patients, the following points should be considered:

- The associations between use of certain antiretroviral therapies with increased CVD are derived from observational data, which can be subject to biases and unmeasured confounding factors.
- The RR for CVD associated with use of certain antiretroviral therapies is small over the short term but may be relevant clinically over longer periods of exposure.
- Some degree of increased CVD risk can be attributed to cumulative metabolic effects (such as dyslipidemia).
- Absolute risk associated with antiretroviral therapy is most clinically relevant in patients with moderate or high CHD risk.

The guiding principle in considering CHD risk in HIV-infected patients is to first maintain control of the HIV infection. Metabolic and other risk factors can and should be managed without compromising treatment for HIV infection.

Coronary Heart Disease Risk Reduction: Managing Dyslipidemia

In CHD risk assessment and management, a patient's absolute CHD risk determines the intensity of risk-reducing interventions. CHD risk is assessed in 3 steps: evaluating for the presence of CHD or risk equivalents (ie, for stroke or transient ischemic attack, peripheral artery disease, and/or diabetes mellitus); counting risk factors; and using

the Framingham risk assessment if 2 or more risk factors are present. The absolute risk determined by the risk assessment process is used to set goals for lipid-lowering and other preventive interventions.

The current National Cholesterol Education Panel goals for lipid-lowering therapy are summarized in Table 1 (Grundy et al, *Circulation*, 2004). The primary target of lipid-lowering therapy is the low-density lipoprotein cholesterol (LDL-C) level; although the target level is based on risk assessment, a LDL-C level below 100 mg/dL is optimal for everyone. The categorical CHD risk factors that modify LDL-C goals include age (men \geq 45 years; women \geq 55 years), family history of premature CHD (ie, CHD in a male or female first-degree relative $<$ 55 or 65 years of age, respectively), cigarette smoking, hypertension (BP \geq 140/90 mm Hg or taking antihypertensive medication), and low HDL-C level ($<$ 40 mg/dL).

Triglycerides (TG) are a primary target of lipid-lowering therapy only when levels are greater than 500 mg/dL because such levels pose an increased risk of pancreatitis. Hypertriglyceridemia is associated with other coronary risk factors (eg, low HDL-C level, hypertension, and insulin resistance) and is mechanistically linked with increased levels of atherogenic remnant lipopro-

teins and the presence of small, dense low-density lipoprotein (LDL) particles. Meta-analyses have shown that hypertriglyceridemia is an independent risk factor for CVD, but it is not as strong a predictor of CVD as are other categorical risk factors.

The non-HDL-C level is a secondary lipid-lowering target when the TG level is greater than 200 mg/dL. Non-HDL-C level is equal to the level of total cholesterol minus the level of HDL-C, and it serves as a measure of all cholesterol present in atherogenic lipoproteins including LDL, very-low-density lipoprotein (VLDL), intermediate-density lipoprotein, and lipoprotein(a). The non-HDL-C level is strongly correlated with the concentration of LDL and apolipoprotein B-100 particles (although not equivalent as a risk predictor) and is a better predictor of CVD events than is LDL-C level. Calculation of the non-HDL-C level does not contain assumptions about the relationship between TG and VLDL cholesterol (eg, as in the Friedewald equation). The target level for non-HDL-C in lipid-lowering therapy is equal to the LDL-C target plus 30 mg/dL.

CHD risk assessment for HIV-infected patients is similar to that for the general population. Patients should have lipid levels measured before starting antiretroviral therapy, and mea-

Table 1. Adult Low-Density Lipoprotein Cholesterol (LDL-C) Level Goals Established by the National Cholesterol Education Panel, Adult Treatment Panel III

Coronary Heart Disease Risk Category	Features	LDL-C Level Goal	Consider Drug Treatment if LDL-C Level Is: ^b
High ("Very high")	Coronary heart disease or risk equivalent ^a	$<$ 100 mg/dL (optional $<$ 70 mg/dL)	\geq 100 mg/dL
Moderately high	\geq 2 risk factors (10-year risk, 10%–20%)	$<$ 130 mg/dL	\geq 130 mg/dL
Moderate	\geq 2 risk factors (10-year risk $<$ 10%)	$<$ 130 mg/dL	\geq 160 mg/dL
Low	0–1 risk factor	$<$ 160 mg/dL	\geq 190 mg/dL

^aCoronary risk equivalent is 10-year risk $>$ 20% or presence of diabetes mellitus, history of stroke or transient ischemia, or presence of peripheral arterial disease.

^bConsider drug options if the level is below the listed goal but above the goal for the next higher risk level.

Adapted from Grundy et al, *Circulation*, 2004.

surements should be repeated every 3 months to 6 months thereafter. After risk assessment, interventions should be undertaken for modifiable nonlipid risk factors, including smoking and adverse dietary habits. If lipid goals are not met with such interventions, lipid-lowering drugs or modification of antiretroviral therapy should be considered. Initial lipid-lowering therapy consists of a statin if LDL-C or non-HDL-C level is elevated and TG level is less than 500 mg/dL, and a fibrate if the TG level is 500 mg/dL or greater. The general sequencing of lipid-lowering therapy for patients not achieving lipid goals with single-drug treatment is shown in Table 2.

Statin treatment is remarkably effective and safe in reducing CVD risk. A meta-analysis by the Cholesterol Treatment Trialists, which included 90,056 patients from 14 randomized statin trials between 1994 and 2004, showed that over a mean follow-up period of 5 years, each 39 mg/dL reduction in LDL-C level with statin treatment was associated with statistically significant reductions of 12% in all-cause mortality, 19% in coronary mortality, 23% in MI or CHD death, 24% in percutaneous coronary interventions or coronary artery bypass grafting, and 17% in stroke (the preventive benefit was for ischemic stroke only) (Baigent et al, *Lancet*, 2005). Approximate dose-equivalence values for available statins are listed in Table 3. Adverse effects associated with these drugs are generally dose dependent.

With regard to the effects of statins in patients with HIV infection, 8 weeks

of pravastatin therapy in individuals receiving antiretroviral therapy was associated with improvement in endothelial function (Hürlimann et al, *Heart*, 2006; Stein et al, *Am Heart J*, 2004). Data are lacking, however, on the prevention of CVD events with statin therapy for HIV-infected patients. Thus far, the best-studied statins for HIV-infected patients are atorvastatin and pravastatin. For patients receiving antiretroviral or other medications that inhibit cytochrome P450 3A4, lovastatin and simvastatin should be avoided, and atorvastatin should be used with caution. Pravastatin is not a very potent statin for lowering LDL-C level, and pravastatin serum levels are increased by concomitant use of darunavir. Rosuvastatin levels are increased by concomitant use of lopinavir/r, but its potency is reduced; it should be used with caution for Asian patients and patients with advanced kidney disease.

For patients not achieving desired reductions in LDL-C and non-HDL-C levels with starting doses of a statin, the first steps are to increase the statin dose and revisit lifestyle interventions. With regard to subsequent options, bile-acid sequestrants have not been evaluated in HIV-infected patients. Extended-release niacin has been used mainly to raise HDL-C and lower TG levels in HIV-infected patients. Ezetimibe has weak effects in lowering

Table 3. Approximate Dose Equivalence of Available Statins for Reduction of Low-Density Lipoprotein Cholesterol Levels

Drug	Dose Equivalence (mg)				
	5	10	20	40	80
Atorvastatin	5	10	20	40	80
Fluvastatin	20	40	80		
Lovastatin	10	20	40	80	
Pravastatin	20	20	40	80	
Rosuvastatin		5	10	20	40
Simvastatin	5	10	20	40	

Adapted from Stein and McBride, University of Wisconsin et al, 2008.

LDL-C and minimal effects in improving HDL-C and TG levels.

With regard to treating hypertriglyceridemia, LDL-C and non-HDL-C levels should be targeted to reduce the CVD risk for patients with TG levels less than 500 mg/dL. For higher TG levels, as noted above, TG level should be targeted to prevent pancreatitis and to assist in lowering the non-HDL-C level. Dietary changes have dramatic effects on TG levels; effective changes including restriction of saturated fats and trans fats, increased consumption of omega-3 and monounsaturated fats, reduced consumption of simple carbohydrates and calories, and restriction of alcohol consumption.

Treatment of hypertriglyceridemia or combined dyslipidemia usually requires combination therapy. Statins have the best evidence for prevention of CVD and a good safety record. Niacin has good evidence for CVD prevention and is safe to use with statins; it is, however, associated with adverse effects that require management. Fish oils have less evidence for CVD prevention; they are safe to use with statins but have a high pill burden. Among fibrates, gemfibrozil has good evidence for CVD prevention. Although fenofibrate has an uncertain effect on CVD risk, it is safer than gemfibrozil to use in combination with statins. Fibrates are the initial treatment option for very high TG levels (> 1000 mg/dL). It is important to treat insulin resistance in patients with ele-

Table 2. General Sequencing of Lipid-Lowering Therapy Based on Triglycerides Level

Medication Sequence	Triglycerides Level		
	< 200 mg/dL	200–499 mg/dL	≥ 500 mg/dL
Initial Treatment	Statin	Statin or niacin	Niacin, fish oils, or fibrate
Second-Line Therapy	Add niacin or bile-acid sequestrant	Statin + niacin	Combination of niacin, fish oils, or fibrate
Third-Line Therapy	Add niacin, bile-acid sequestrant, or ezetimibe	Add ezetimibe	Add third triglyceride-lowering drug, consider adding statin

Adapted from Stein and McBride, University of Wisconsin Health et al, 2008.

vated TG levels and combined dyslipidemia, with treatment including diet and exercise modifications and medications (eg, metformin or pioglitazone).

Lecture presented by Dr Stein in June 2010. First draft prepared from transcripts by Matthew Stenger. Reviewed and edited by Dr Stein in November 2010.

Financial Disclosure: Relevant to this article, Dr Stein has received subcontracted research support from the University of North Carolina that originated from GlaxoSmithKline. He has been a member of data and safety monitoring boards for Abbott Laboratories, Eli Lilly and Co, and Takeda Pharmaceutical Co, Ltd.

Suggested Reading

Baigent C, Keech A, Kearney PM, et al. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90,056 participants in 14 randomised trials of statins. *Lancet*. 2005;366:1267-1278.

Bozzette SA, Ake CF, Tam HK, Chang SW, Louis TA. Cardiovascular and cerebrovascular events in patients treated for human immunodeficiency virus infection. *N Engl J Med*. 2003;348:702-710.

D:A:D Study Group, Friis-Møller N, Reiss P, et al. Class of antiretroviral drugs and the risk of myocardial infarction. *N Engl J Med*. 2007;356:1723-1735.

Greenland P, Knoll MD, Stamler J, et al. Major risk factors as antecedents of fatal and non-

fatal coronary heart disease events. *JAMA*. 2003;290:891-897.

Grundy SM, Cleeman JI, Merz CN, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. *Circulation*. 2004;110:227-239.

Hürlimann D, Chenevard R, Ruschitzka F, et al. Effects of statins on endothelial function and lipid profile in HIV infected persons receiving protease inhibitor-containing anti-retroviral combination therapy: a randomised double blind crossover trial. *Heart*. 2006;92:110-112.

Lundgren J, Reiss P, Worm S, et al. Risk of myocardial infarction with exposure to specific ARV from the PI, NNRTI, and NRTI drug classes: the D:A:D study. [Abstract 44LB.] 16th Conference on Retroviruses and Opportunistic Infections (CROI). February 8-11, 2009; Montreal, Canada.

Phillips AN, Carr A, Neuhaus J, et al. Interruption of antiretroviral therapy and risk of cardiovascular disease in persons with HIV-1 infection: exploratory analyses from the SMART trial. *Antivir Ther*. 2008;13:177-187.

Stein JH and McBride PE. 2008 guidelines for the diagnosis and management of dyslipidemia for adults ≥ 18 years of age, May 2008. University of Wisconsin Health Physicians Plus Insurance Corp, Group Health Cooperative, Unity Health Insurance. www.pplusic.com/uploads/media/LIM13973DyslipidemiaCG.pdf. Accessed November 8, 2010.

Stein JH, Merwood MA, Bellehumeur JL, et al. Effects of pravastatin on lipoproteins and endothelial function in patients receiving hu-

man immunodeficiency virus protease inhibitors. *Am Heart J*. 2004;147:E18.

Strategies for Management of Antiretroviral Therapy (SMART) Study Group, El-Sadr WM, Lundgren JD, et al. CD4+ count-guided interruption of antiretroviral treatment. *N Engl J Med*. 2006;355:2283-2296.

Strategies for Management of Antiretroviral Therapy/INSIGHT, D:A:D Study Groups. Use of nucleoside reverse transcriptase inhibitors and risk of myocardial infarction in HIV-infected patients. *AIDS*. 2008;22:F17-F24.

Torriani FJ, Komarow L, Parker RA, et al. Endothelial function in human immunodeficiency virus-infected antiretroviral-naive subjects before and after starting potent antiretroviral therapy: The ACTG (AIDS Clinical Trials Group) Study 5152s. *J Am Coll Cardiol*. 2008;52:569-576.

Worm SW, Sabin C, Weber R, et al. Risk of myocardial infarction in patients with HIV infection exposed to specific individual antiretroviral drugs from the 3 major drug classes: the data collection on adverse events of anti-HIV drugs (D:A:D) study. *J Infect Dis*. 2010;201:318-330.

Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*. 2004;364:937-952.

Top HIV Med. 2010;18(5):164-168

©2010, International AIDS Society–USA

Topics in HIV Medicine®

Continuing Medical Education

The following article in this issue is associated with CME credit:
Stein JH. Evaluating and managing cardiovascular disease risk factors in HIV-infected patients.
Top HIV Med. 2010;18(5):164-168

Instructions

This Continuing Medical Education (CME) activity provides a review of data regarding the management of cardiovascular disease risk factors in patients with HIV infection. To complete the activity, the learner is instructed to:

- Read the article
- Review a selection of the references
- Reflect on how the information might be applied to the clinical practice
- Take the posttest
- Complete the CME claim form and send it to the IAS–USA office.

Objectives

Upon completion of this activity, learners will be able to describe results of recent research and the potential clinical implications for their HIV-infected patients on the evaluation and management of cardiovascular disease risk factors.

Accreditation Statement

The International AIDS Society–USA is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

The International AIDS Society–USA designates this activity for a maximum of 1.5 *AMA PRA Category 1 Credits™*. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Intended Audience

This activity is intended for physicians involved in the care of patients with HIV infection. It is also relevant to nurse practitioners, physician assistants, nurses, and other health professionals who provide care for people with HIV disease.

Author Financial Disclosures

Dr Stein has received subcontracted research support from the University of North Carolina that originated from GlaxoSmithKline and has been a member of data and safety monitoring boards for Abbott Laboratories, Eli Lilly and Co, and Takeda Pharmaceutical Co, Ltd.

Posttest Questions

Circle the single best answer to each of the questions below.

1. In the INTERHEART study, modifiable risk factors accounted for what percent of the population-attributable risk for myocardial infarction?
 - A. 21%
 - B. 42%
 - C. 63%
 - D. 84%
2. In the D:A:D study, use of which of the following protease inhibitors was associated with a statistically significant increased yearly risk of myocardial infarction?
 - A. Lopinavir/ritonavir
 - B. Saquinavir
 - C. Nelfinavir
 - D. Atazanavir
3. In the D:A:D study, use of which of the following nucleoside analogue reverse transcriptase inhibitors was associated with a statistically significant increased yearly risk of myocardial infarction?
 - A. Lamivudine
 - B. Stavudine
 - C. Zalcitabine
 - D. Abacavir
4. What are the target low-density lipoprotein cholesterol (LDL-C) and non-high-density lipoprotein cholesterol (HDL-C) levels for patients with coronary artery disease?
 - A. LDL-C < 100 mg/dL, non-HDL-C < 100 mg/dL
 - B. LDL-C < 100 mg/dL, non-HDL-C < 130 mg/dL
 - C. LDL-C < 130 mg/dL, non-HDL-C < 100 mg/dL
 - D. LDL-C < 130 mg/dL, non-HDL-C < 160 mg/dL
5. Regarding treatment of dyslipidemias, which of the following statements is correct?
 - A. Statin drugs are the preferred initial treatment for all patients
 - B. Pravastatin has no drug interactions with HIV treatment medications
 - C. Statin drugs, niacin, or fenofibrate are preferred treatment strategies for patients with triglycerides levels above 500 mg/dL
 - D. Statin drugs have the best evidence base for prevention of cardiovascular disease

This CME activity is offered from **January 15, 2011**, to **January 15, 2012**. Participants who complete the activity posttest and submit the registration form are eligible to receive credit. Physicians (MDs, DOs, and international equivalents) may receive CME credit for completing this activity. Nonphysician health care practitioners will receive a certificate of attendance.

Please complete and return this form to receive CME credit

Participant Information

Number of CME credits I am claiming (maximum 1.5): ____

First Name _____ MI _____ Last Name _____

Telephone (____Home ____Work) _____ Facsimile _____

Degree or License (MD, RN, PA, none, etc) _____

E-mail address to receive CME certificate (____Home ____Work) _____

Institution or Organization _____

The amount of time (in hours) I spent on reading the article, reviewing the references, reflecting on how the information might be applied to the practice, and taking the posttest was:

Address (Please check one: ____ Home ____ Work) _____

≤ 1 1.5 2 3 other _____

Address 2nd Line _____

Fax or mail this form to:
International AIDS Society–USA
425 California Street, Suite 1450
San Francisco, CA 94104-2120
Fax: (415) 544-9401

City _____ State/Province _____

Postal Code _____ Country _____

Correction

There was an error in the printed version of *Topics in HIV Medicine*, volume 18, issue 3, in the article “Dyslipidemia and its Treatment in HIV Infection.” On page 116, column 2, paragraph 1, “fosamprenavir/r” in line 9 was inadvertently switched with “atazanavir/r” in line 17 below. The text should have read:

Rosuvastatin maximum concentration was increased 4.7-fold by lopinavir/r (Kiser et al, *J AIDS*, 2008) and 6-fold by atazanavir/r (Busti et al, *J Cardiovasc Pharmacol*, 2008). Thus, high-dose rosuvastatin should be avoided by patients receiving these PIs. No change in rosuvastatin concentration was observed with fosamprenavir/r (Busti et al, *J Cardiovasc Pharmacol*, 2008).

The correction has been made in the online version of the issue at <http://www.iasusa.org/pub/topics/2010/issue3/112.pdf>. In addition, the same error has been corrected in a keyslide from the original course presentation (presentation 2, slide 24) available at <http://www.iasusa.org/keyslides/2010/slosangeles/index.html>.